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THE

PSYCHOLOGICAL BULLETIN

GENERAL REVIEWS AND SUMMARIES

THE FUNCTIONS OF THE CEREBRUM[1]

BY SHEPHERD IVORY FRANZ

Government Hospital for the Insane

One of the good results to be expected from the present great war is that of advancement of our knowledge regarding the functions and functional relations of the parts of the nervous system. Already indications are at hand that much attention is being directed to the effects of injuries of peripheral nerves, to sections of or disturbances with the spinal cord, and to destructions of and the effects of "shock" upon parts of the cerebellum and the cerebrum. The conditions of warfare are such that head injuries from bullets and from shrapnel are common and many of these include parts of the brain. The organization of the medical corps, the governmental and private training schools for cripples and for others who are unfitted to undertake their former occupations, and the neurological societies, although not necessarily coöperatively, will investigate those who survive and we shall have from these studies more facts regarding the injurious effects of cerebral lesions in man, and subsequent recoveries or consequent defects, than have been accumulated in many years. The recent medical journals contain many clinical accounts of lesions of the brain or other parts of the nervous system (see especially **15**, **21**, **34**, **41**, **42**, **44**, **47**) and the attention of physicians is being directed to them more and more on account of the resulting permanent defects. No such extensive and systematic study as those of Head and his co-workers on the peripheral nerves

[1] For previous reviews see PSYCHOL. BULL., 1911, 8, 111–119; 1913, 10, 125–138; 1914, 11, 131–140.

has yet appeared, but much promise of such contributions is given by the articles of Poppelreuter (42) and of Marburg and Ranzi (34), by the suggestions of Fröschels (21) and by the protest of the Neurological Society of Paris to the department of war that neurological cases are not sent sufficiently early to specialists for careful neurological and mental examinations.

General.—Anatomists have been divided with respect to the reason for the formation of the fissures of the brain. Some have held that they were produced by growth antagonism, *i. e.*, by the limited cranial capacity and the increase in the growth of the cells and fibers, while others have contended that the fissures were due to local differentiation of parts. Jefferson (24) shows that both factors are probably at work, although not necessarily in the same brain at the same time, for he brings forward as evidence opposed to the local differentiation view the fact that there is a considerable amount of areal differentiation in the brains of some monkeys (*Hapale jaccus* and *Lemur niger*) but there are few fissures. On the other hand, although careful maps of cyto- and myelo-architecture have not been made, it appears probable that not as great differentiation in structure has taken place in the cortices of cetacea and the elephant as in man, but the brains of the elephant and some cetacea have more furrows than are found in man and in the primates in general. In these cases, therefore, it appears probable that growth antagonism is the more important factor in fissure formation. On the other hand, Jefferson has also shown that when "furrows do appear they tend to do so at the edges of specialized areas," and he concludes that "we must look to architectural changes in the gray matter as the guiding influence in the *placing* of the furrows."

This question in some of its other relations has been discussed by Mills (36). He has pointed out that fissures have a certain amount of physiological significance not only in the cerebrum but also in the cerebellum. Mills's article does not lend itself to brief summarizing, and the reader is referred to it for the discussions, not only on the topic mentioned but for his consideration of neurobiotaxis, cytological anatomy and morphology, the cell type localization in the optic thalamus, and histological anatomy and morphology in relation to mental diseases.

Dogs with the hemispheres completely removed, similar to the brainless dog of Goltz, have been described by Zeliony (56). Two of these lived respectively only three and four days, a third lived

over eleven months, and a fourth was living and shown to a society nearly sixteen months after the removal of both hemispheres. The dogs walked freely but showed considerable ataxia and in walking knocked against objects in the room; food when placed in the mouth was swallowed; sounds even of small intensity produced characteristic pricking of the ears (Goltz's dog reacted only to loud sounds and then only with head movements); the taste reactions were normal in that food caused a secretion of saliva and when it contained quinine it was rejected; light stimuli produced pupillary movements and the animal turned its head in the direction of the stimulus; but no conditioned reflexes were produced. The autopsies showed that the amount of cerebrum removed in all cases exceeded that removed by Goltz from the dog operated upon by him. These results show how much of the cerebrum may be removed without affecting very greatly the capability of the animal to react to ordinary forms of stimuli, although the animals were not very active and did not have that thing called "initiative" which is characteristic of animals with the cerebrum intact.

The difficulties attending the removal of large pieces of the cerebrum in dogs are very great, but in monkeys much greater. The complete removal of both hemispheres of the monkey has been accomplished by Karplus and Kreidl (**27**) with results of very great interest. Seventeen monkeys were (surgically) successfully operated upon, the animals having one hemisphere removed at one operation and the second at a subsequent session, from ten days to eleven months intervening. Many of the animals lived for only very brief periods, one day or less, but some lived for more than a week and one as long as twenty-six days after the removal of the second hemisphere. In the account of the work the authors give illustrations of the appearance of two animals with one hemisphere removed and of one animal with both hemispheres extirpated. Reproductions of microscopical sections of the brains of eight monkeys are also given. In a later communication Karplus (**26**) considers the degenerations in the pyramidal tracts of the spinal cord and the medulla oblongata in these animals.

The following conditions were found by Karplus and Kreidl in the monkeys from which one hemisphere had been removed, all the defects being located on the side opposite to the lesion. The animals sat upright in their cages, they ate food, climbed upon the bars of the cages, and performed other acts almost like a normal animal. An hour after the operation one animal was seen to brush

away a fly which had alighted upon it. Immediately after the operation there was a marked degree of weakness in the contralateral extremities, which was more evident for the anterior than for the posterior segment. After the immediate effects of the operation had worn off so that casual observations failed to indicate defects, there were difficulties in grasping food and the movements of the hand and fingers were not as accurate as those on the normal side, and the movements of the distal segments were less normal than those at the elbow and shoulder. The capability of moving the head was apparently not disturbed. No convulsions were observed. In some cases when the sound side was moved there was also a contralateral movement (*i. e.*, of the affected side). There was no observed general mental change, but there was permanent hemianopsia, and an apparent contralateral hypesthesia although the animals reacted definitely to strong painful and temperature stimuli. No disturbances of the eye muscles were found except that there was a temporary nystagmus in two animals, and the animals appeared to hear and to discriminate sounds as well as usual for they reacted to the barking of dogs and to the chattering of other monkeys. No difference in effect of removing the right or the left hemisphere was observed, except that of the location of the defect on the contralateral sides.

These animals recovered to a great extent their ability to move the side affected by the first operation, and after the removal of the second hemisphere it was found that the side first affected was moved more than the newly affected side. In the animals lacking all of the cerebral cortex movements of the head and eyes were not disturbed but movements of all extremities were greatly affected; a kind of athetoid movement of the arm was made by some of the animals more than 100 times in succession. The day following the removal of the second hemisphere from one animal it took hold of the bars of the cage with the hand first paralyzed (because of removal of the first hemisphere) and pulled itself up into a sitting posture which it held for some time. In four cases tonic contractions of the extremities were noticed, in two cases there were clonic spasms, and one animal showed what appeared to be an intention tremor. When stimulated on the skin the animals lifted their heads, opened their eyelids, and there was dilation of the pupils. When sounds were made the ear movement reflexes were produced and at the same time there were eyelid movements. Light stimuli provoked pupillary constriction; two animals showed nystagmus.

The animals could cry as usual, and they did cry especially to painful stimuli, but there were no countenance changes coincident with the reflex responses. Most of the animals made no voluntary movements during the first days after the removal of the second hemisphere and they had their eyelids closed and reacted only slightly to stimuli. There appeared to be a kind of sleepy condition which could be changed to the waking-like state by strong stimuli. The great difficulty in feeding the animals probably was a factor in causing the death of some.

Not only because of the difficulty of the operative technique are these results of interest, but they have a definite interest in showing plainly that there are no real differences between the activities of the basal ganglia in the monkeys and in the lower animals, and that life can be sustained with the whole of the cerebral cortex removed in the so-called higher animals. They show also that the cerbral cortex is not nearly as important in the carrying out of some of the ordinary activities of an animal as has been supposed, and there is an indication that, as will also be shown below, the cortical mechanisms are not necessary for many of those functions which have been assumed by it.

This is confirmed by the results of the comparison of the brain of the patient examined by Robinson (**43**) with the clinical symptoms. This patient exhibited a slight enfeeblement of "intelligence" and some diminution in memory ability, while hearing, taste, and touch were almost normal and speech was scarcely interfered with. Vision was gradually lost. The brain showed a necrobiosis almost as far as the central fissure without any of the notable symptoms which have been supposed to be associated with lesions of the frontal portion of the cerebrum, and the author believes that the lack of symptoms are to be accounted for on the ground of compensatory processes, some parts of the cerebrum assuming functions normally undertaken by other parts when the latter are diseased. This would be found in those cases in which the pathological process was slow and there was sufficient time for the compensation.

That the definiteness of function is not absolute is well shown by the cases of nerve anastomosis which will be discussed below, but in this connection mention may be made of the results obtained by Claude, Dumas, and Porak (**12**) in cases in which paralyses of certain muscles exist, but there is a compensation by the utilization of other muscles which are not ordinarily, or strongly, used for the production of the kinds of movements normally brought about by those

which were paralyzed. It is apparent that some possibility of functional adaptation exists in the brain for certain types of movement so that when a certain "center" and its connected muscles cannot be utilized other "centers" and their connecting muscles may be brought into play to bring about the desired result. This view is at the basis of the work of Kouindjy (29) who has by reëducation brought about a restoration of function in such conditions as tabes and astasia abasia, and who also mentions as a possibility the reëducation of paralytics.

Of the general effects following distrubances of the cerebrum Poppelreuter (42) and Marburg and Ranzi (34) have given accounts. Poppelreuter examined thirty patients with brain injuries and found not only the local effects which have been associated with localized lesions, but also general effects upon the "personality" of the individuals. In some cases the defects were almost psychological localizations (as distinct from cerebral-mental localizations). Thus, he reports the case of a student who had forgotten all chemical formulas, and another patient who could not do sums in division although there was no apparent aphasia and no other disturbances of the mental processes were evident.

Rothmann (44) has utilized a number of cerebral accidents to soldiers to point out that our experimental results on brain physiology of animals are being confirmed in man, and that the experimental facts point the way to diagnoses most clearly.

The works of both Krasnogorski and of Brown deal with the development of cortical cerebral control. That of Krasnogorski (31) considers the conditional reflexes in children and especially the development of this mode of reaction. He has found that these reflexes in children are essentially the same as in animals, although they differ in that they are rapidly formed and are much more stable, but may be readily replaced. He has found that they develop in the second half of the first year of life and that they are more apparent in the second year. He has expressed the belief that by the use of the conditional reflex test it is "possible to follow the entire development and gradual complication of the cortical activity in childhood, from the first few weeks of life, but it is also possible to recognize even in the first few months of life the characteristics and defects in functions of the cerebral cortex, and to apply as soon as possible the corresponding therapeutic measures."

Cortical Motor Control.—An extension of the investigation of the localization of the motor centers of the monkey brain has been made

by Franz (**18**, part 2) in which he has shown that there are considerable differences in the spatial localization of the motor areas in the brains of different animals and in the two hemispheres of the same animal. The leg areas in some animals are larger than and in some smaller than the arm areas; there are considerable variations in the areal amounts concerned with the movements of different segments of the larger areas, for example for the toe and wrist movements; the areas for the arm and leg segments overlap to a great degree so that combined movements of both segments are normally obtained in some animals; a number of non-stimulable or relatively non-stimulable zones lying within stimulable areas were found. These results, with others from other observers, have led to the formulation of an hypothesis regarding the variability of function of different brains and its relation to differences of behavior. All of this work indicates that the motor areas lie in front of the central fissure. This is contrary to the view expressed by the late Professor Rothmann (**45**) who has continued to follow the conclusions of Munk, and who was the only recent well-known neurologist to adhere to the latter's views. Amantea (**1**) also upholds this view. Rothmann, working with dogs, found what he considered to be a zone for deep sensibility behind Munk's leg region, for the foreleg in the anterior suprasylvian gyrus and for the hind limb in the anterior part of the marginal gyrus. Destruction of these areas brought about derangements of the opposite extremities in all directions. In monkeys he found that destructions of the supramarginal gyrus produced difficulty of movement, of the nature of an ataxia, of the opposite arm, but no eye movement disorder. The extirpation of the postcentral region also produced motor disturbances of the opposite side, like an ataxia. At the same time the opposite arm was little used, although it could be used. He concludes that the "inactivity of the opposite arm after destruction of the postcentral gyrus as well as the restitution of isolated movements of the arm after destruction of the precentral gyrus shows the existence of motor elements in the cortex of the postcentral gyrus."

Brown has dealt with some of the more general conditions accompanying the activity of the cortex. Whether or not the act of walking is a learned act is a question the answer to which may help towards the solution of other similar problems. This Brown has taken up (**9**) in experiments upon the unborn fœtus of the cat. When the unborn fœtus is shelled out of the uterus into warm normal salt solution it sometimes shows movements of progression

which are apparently spontaneous. The neural mechanisms are thus found to be able to function before birth, and the fact that such complex movements may be produced is evidence that walking is not a learned act which at a subsequent period becomes automatic or reflex. It appears that it is primarily a reflex activity, and that this activity may be first found in intrauterine life. The results of this study would serve to indicate that certain complex acts which we have been accustomed to believe must be learned by trial and error methods are not of that character, but that they may be only expressions of the physiological combinations of neural mechanisms which require for the activity a particular stimulus at a certain time. This is what is found to be true for the act of respiration. Brown has also (11) shown that if an electric stimulus be applied to a motor area and after the reaction is obtained it be applied again there is an increased result at the second application. The "facilitation" lasted for about ten seconds after a one-second stimulation, but after ten seconds there was no summation effect. This fact is of interest in connection with certain problems of motor learning.

Nerve Anastomosis and Recovery of Motor Function.—One of the most interesting topics in cerebral localization is that connected with the recovery of function after nerve anastomosis. It has been found that it is possible so to cross-join nerves which have been cut that complete motor recovery takes place. The fibers regenerate and although the central paths must have been altered there is a complete return of voluntary control, and, more remarkable, of mimetic movements in the segment involved. Operations of this character are not infrequently performed at the present time for those individuals who have lost, because of nerve lesions, certain important motor functions and the effort is made to take as a "leader" a nerve which has less important functions and join it to the peripheral end of the more important nerve which has become functionless. This has been done by Welty (53) who has described a case in which he used the central end of the hypoglossal nerve to join to the peripheral end of the facial nerve. At the end of a year the patient could move the face muscles, and three years after the operation it was found that the side of the face which was paralyzed was moved when the patient talked, the patient could close the eye, wrinkle the face, and lift the eyebrow. Some atrophy of the tongue was present on account of the use of the hypoglossal nerve and this probably also brought about the slight defect of speech which was

noticed. In such cases of facial paralyses it has been more usual to use the spinal accessory nerve which innervates the shoulder muscles, the resulting paralysis of the shoulder being usually less important than the facial paralysis.

The experiments of Kennedy (**28**) have given us some fundamental facts regarding the conditions in nerve anastomosis in animals which may be applied directly to man. He found that he could cross the musculo-spiral nerve with the musculo-cutaneous, the median and the ulnar nerves, but at the same time he has been able to demonstrate that one nerve may be used for all of the functions involved. If, however, only one nerve (or set of nerves) with one original function is used the return of function of that character (for example, flexion) is quicker than that of the opposite function (extension). At the same time he has shown that if no nerve be eliminated the return of function is quicker. If the flexor nerve has been eliminated and the cortex area for flexion be stimulated after the return of voluntary function no response is obtained, and vice versa for the extension. Here, by way of an aside, it may be asked how the author has arrived at the conclusion that a certain spatially located point is the flexion center. In view of the normal reversability of action of the centers, as shown by Sherrington and Brown, and in view of the differences in distribution of the motor areas, as shown by Franz, an answer to this question is of the greatest importance if we are to accept some of the resulting theoretical conclusions. Kennedy believes that in the functional return there is a greater or less amount of reëducation of the cortical centers, such reëducation being related to or dependent upon the afferent impulses from the appropriate tissues. The adaptation of the cerebral centers is, however, not considered by Kennedy to be due entirely to a kind of reëducation, because in some of the animals there was no possibility of practice on certain of the movements, on account of the artificial limitation of these movements by means of plaster casts, bandages, etc. He believes that the recovery is due, partly at least, to "an alteration in the centers under the influence of altered afferent impulses from the muscles, the brain thus having the capacity quickly to adapt itself to such alteration," an explanation which appears to the reviewer to be subsumed under the general term reëducation.

The possibility of regeneration of nerves and function when two nerves of the same general kind, afferent or efferent, are used is well known at the present time. It has been disputed that there is any

cross-recovery when an afferent nerve is joined to an efferent. The possibility of this kind of regeneration has been apparently demonstrated by Boeke (5), who united the peripheral cut end of the lingual nerve (afferent) to the central cut end of the hypoglossal nerve (efferent). After from two to five months Boeke tested for the return of motor power in the tongue and found this to be present. It should be noted, however, that neither of these two nerves can be considered to be "pure" in the sense that they respectively convey only afferent and efferent impulses, but the return of function is nevertheless noteworthy as indicating the possibility of such afferent-efferent junctions.

Closely allied to the subject of nerve anastomosis is that of muscle splitting. Kennedy (28) cites the case of a young girl with talipes equinus in whose leg the gastrocnemius was split, and part of the muscle was brought to the anterior part of the leg to replace the degenerated and functionless flexors which included the tibialis anticus. The transplanted part was found to be used for flexion as the other, undisturbed, part for the normal extension of the leg. In such cases Kennedy considers there is the same kind of adaptation in the cerebrum as in nerve anastomosis. Such an admission of the creation of new "centers" in the cerebrum goes far towards breaking down the uncritically-believed views of cerebral function. The compensations noted by Claude, Dumas, and Porak (12) should also be remembered.

Recovery of Motor Function after Cerebral Paralysis.—Although closely allied to the recoveries of motor function after nerve anastomosis those which follow the loss of the cerebral motor cortex or the destruction of the conduction fibers differ from the former in many ways. The possibility of recovery of this kind in animals has been recognized for a number of years. Trendelenburg (51) returns to the general problem, using monkeys, and he finds that although an animal loses its ability of voluntary movement immediately after the cortical ablation, movements of certain kinds may still be present. Thus, when the cortical area for the right arm was destroyed the right arm could be used for climbing, but the left alone was used for eating, but in about four weeks the animal began to use the right arm for the finer movements. When the right motor cortex was stimulated movements on the left were produced but none on the right. This shows that the recovery has not been due to an assumption of bilateral innervation by the sound side of the brain. It was also found that if the normal arm was

restrained the paralyzed arm was moved more than if both arms were free. The movements which were produced were awkward but an adjustment could be made. There was not an actual inability to move but what appeared to be a disinclination to move. The dogs operated upon by Zeliony (**56**) and the monkeys of Karplus and Kreidl (**27**) also showed considerable ability to move, even though one or both hemispheres were taken away.

In man it has been known that a certain amount of recovery may take place within a brief period after such a cerebral accident as a hemorrhage, and an initial complete hemiplegia may give way to an incomplete paralysis. It has been thought, however, that the destruction of certain cerebral cells or their fibers would result in an absolute paralysis of certain parts. Several recent investigations show that this is not the case. Poppelreuter (**42**), for example, has had good success in the reëducation of paralytics with recent lesions. He has shown that systematic training gives very gratifying results in that movements supposedly forever gone have returned and the patients have learned to use such an instrument as the typewriter. Kouindjy (**29**) also has shown that reëducation is of value in cases of paralysis. The cases with which Poppelreuter and Kouindjy worked were recent cases; those with whom Mayer worked were two cases respectively of nine months and of two years' standing. Mayer (**35**) found that orthopedic treatment was beneficial in these two cases, although the most that resulted was the correction of the deformity of contracture which follows the initial motor inability. He obtained a betterment in the gait of his patients and an improvement in other motor functions. The most conclusive work showing that the destruction of cortical areas or of subcortical connections in man does not mean a complete loss of function is that of Franz, Scheetz, and Wilson (**19**). Taking patients who had had paralyses for periods of from five to twenty years they have shown that systematic passive exercises, combined with massage, may result in the return of certain movements, which is contrary to the belief of many neurologists. Most of the motor return was for the gross movements of the body, but it has been shown that coordinated movements such as throwing a ball, using a fan, sewing, etc., could be performed after a period of "training."

The results on man are directly comparable with those on animals, although the lesions in the human "experiments" cannot be as definitely determined as those in animals, without waiting for the natural deaths of the subjects. The human cases of paralysis

have been selected on clinical grounds, and the possibility, although a very rare possibility, of the paralyses not being the result of lesions of the motor cells or fibers in the cerebrum must be kept in mind. Assuming the cerebral conditions to be those which the clinical conditions would suggest, it is possible to conclude that the motor cortex is not essential for the performance of voluntary actions, and although the definite localization of the cells concerned in the recovery has not been made, the suggestion is offered that the lenticular nucleus and other subcortical collections of gray matter may take part in this. If this can be proven, we shall have to accept what has already been hinted at in the past, viz., that the exclusive localization of "consciousness" in the cortical areas is neither not proven nor probable.

In connection with the recoveries of motor function by paralytics the study of Misch and Lotz (**39**) has an interest in that it shows clearly the fact that paralyzed muscles exhibit action currents, and the conclusion is apparent that innervation must proceed from some part of the nervous system other than the cerebral cortex.

As an example of the adaptation of the nervous system, and perhaps especially of the cerebrum, may be cited the results of Claude, Dumas, and Porak (**12**) on the recovery of certain functions when peripheral nerves have been divided. These authors find that even though certain muscles may be completely paralyzed there is a possibility of the performance of many tasks which these muscles usually perform. They find, for example, that the hand can be used even though certain muscles can not be employed in the acts, and that this "recovery" is due to the adaptation of other muscles to the particular ends. This substitution of action is sometimes mistaken for the recuperation of function by a paralyzed muscle. The amount of adaptation that a part may undergo is limited by the paralysis to some extent, but in the other direction only by the intelligence and ingenuity used to overcome the defect. The patients try repeatedly to find a method to overcome their deficiency and finally they usually succeed in finding some position in which a limb may be placed to bring about the desired action. These observations are similar to those of Kennedy (**28**) which have already been reported above, and they show that the adaptations, or new physiological connections, of which the cerebrum may be capable are not of minor importance, but that they are of most frequent occurrence even for the supposed simple motor operations. At the same time they serve to bring home the fact, which has often

been emphasized since the time of Flourens but most frequently lost sight of, that the cerebrum is not a simply organized physiological organ, but one with most complex and intricate arrangements and possibilities of adaptation.

On the negative side of cerebral localization Dupuy (16) takes up those cases of so-called clinical cerebral paralysis without cerebral lesions, and the cases of cerebral motor(?) lesions without paralyses. He believes that most, if not all, of the facts can be explained on the Brown-Séquard hypothesis of inhibition, and this is very close to the more modern conclusions of von Monakow. In almost the same vein Robinson (43) has dealt with some negative cases. He cites one case in particular in which although there was slight "enfeeblement of intelligence" and diminution in memory, there was little or no interference with hearing, taste, touch, and speech, although vision had been gradually lost. At the autopsy the brain was found to be almost entirely destroyed nearly up to the central fissure. Robinson has concluded that there are notable compensations in cerebral activities whenever any part of the brain is destroyed, and these have an opportunity to be utilized especially when the destructive process is slow. This is not unlike those compensations, of whose existence we appear to know much more, between the cerebellum and the cerebrum.

Cortical Epilepsy.—Two studies on the brain states in experimental epilepsy have been made, respectively, by Karplus (25) and by Bikeles and Zbyszewski (4). Karplus attempted to determine the course of the impulses which gave rise to the diffuse convulsions when one hemisphere was stimulated, for contrary to what might a priori be supposed the stimulation of one hemisphere brings about convulsive conditions in both halves of the body. It was formerly believed that the corresponding motor cortex was affected in some manner, perhaps by way of the corpus callosum through which impulses initiated on one side passed to the corresponding areas on the other side. This has been shown not to be always the case, for when the corpus callosum is divided the bilateral convulsions can be produced. Karplus concludes that the bilateral connection is made by way of some fibers in the brain stem, but whether in the medulla oblongata or in the hypothalamus could not be settled. The results obtained by Karplus are not conclusive in that they do not definitely prove that the lower branching of the impulses is the "normal" method, for there are good reasons to believe in the close association of the two precentral (motor)

areas by way of the corpus callosum, and it is known that if one pathway be blocked there is a possibility of using one or more normally little used routes. Bikeles and Zbyszewski have attempted to discover the reason for some of the improvement in epilepsy when different drugs are administered. They have obtained results which indicate that those drugs which help to reduce the convulsions do not always produce a decrease in the irritability of the motor cortex, and the results suggest that investigation of the whole cerebrospinal mechanism is necessary before we shall be able to explain the complex but apparently rhythmical phenomena in the epilepsies.

Subcortical and Subsidiary Motor Functions.—Using as a text the account of a patient whose brain showed lesions of the caudate and lenticular nuclei on the right side and of the caudate and part of the lenticular nuclei on the left side, Mills (37) has considered the relation of the cerebrum and the basal ganglia to muscular tonicity and to spasmodic phenomena. Spasmodic crying and other similar phenomena are considered by him to have relation to the cerebral cortex, not to the lenticular nuclei as others have contended, and he considers these phenomena to be examples of abnormal tonicity. He disputes Luciani's conclusions that the cerebellum is that part of the central nervous system which controls muscular tone, and considers that the midfrontal or intermediate precentral areas are concerned in that function. These regions are connected with the corpus striatum by association fibers, and he would have us believe that the caudate nucleus is like the cerebral cortex, or almost as a part of it. Holmes (23), on the other hand, has contended that the facts can be understood best as the result of the overactivity of subcortical motor centers "when freed from the inhibitory control of the cortex." This is especially true for the spasticity in paralysis. The involuntary movements which have been associated with thalamic lesions are also due to "disturbance of the normal functional equilibrium between the cerebellothalamic and the cortico-spinal systems." A third explanation of the spasticity in paralysis has been offered by Förster (17) who considers that the condition is due to a "sensory charging." Some have believed that the anterior horn cells in the spinal cord are those most concerned in the production of the spasticity, and that they act because of irritation from the pyramidal fibers. This does not help us to understand the fact that spasticity accompanies the state when all pyramidal fibers are destroyed, as in congenital spastic paraplegia, so that it is concluded that the anterior horn cells are "charged" by impulses coming from the periphery.

In the discussion of "tonic innervation" Wilson and Walshe (55) also deal with a similar matter. They have added three cases to the one previously described by Wilson in 1908, which showed tonic perseveration, which is a "condition in which there is an inability, owing to a central lesion, to relax a given innervation in any muscular group or groups." This has also been observed by Liepmann in states of apraxia. The condition is aggravated by attention to the movement, and they consider it to be an inability to "inhibit an innervation" which has been initiated. This is related to impairment of function of the cortico-spinal neurones, and is probably due to interference with "short transcortical paths from a hypothetical psychomotor center placed in front of the precentral gyrus." This, it will be noted, is similar to the explanation offered by Mills.

Southard (49) has presented an interesting account of some hyperkinetic conditions which are associated with thalamic lesions. He has taken cases showing diffuse lesions of the optic thalamus and compared them with other brain lesions with respect to involuntary and, perhaps also, voluntary motor activity. He had found a greater amount of motor activity in the former cases, but he does not believe the motor symptoms are indicative of motor function of the thalamus. In fact, he explains them as the effects of the "withdrawal of corticothalamic 'inhibitory' or 'switch-setting' impulses" or of "atrophy or aplasia of certain cerebellar connections." Here, therefore, is a condition in which a lesion of a kind of afferent mechanism brings about a symptom which appears to be that of the efferent system.

Some have claimed for the striatum a steadying influence on the motor impulses, but Lloyd (33) believes that it has sunk to a position of very slight importance, although at one time in the phylogenetic scale it was of great value. He contends that there is no special syndrome referable to the lenticular nucleus and that all the symptoms which others have referred to disturbances of that body are readily explainable on the ground of involvement of other related structures. He has disputed that the emotional reactions in lenticular cases are directly due to the lenticular nucleus lesions, and he concludes that laughing occurs in these cases only because some cortical fibers are cut off, and that the emotional reaction becomes a reflex. Nammack (40), on the other hand, agrees with the original contentions of Wilson regarding the lenticular syndrome, and reports a case characterized by the symptoms described by that

author: involuntary movements, dysarthria, muscular weakness, spasticity, emotionalism. He accepts the explanation that in that state we deal with a disturbance of the extracortical motor system.

The stimulation of different parts of the basal ganglia by Brown has given results of interest in this connection. He used a chimpanzee about 30 months old. When happy this animal protruded its lips and gave a cry like "Ou, Ou, Ou," and when left alone it whimpered or moaned like a child (but did not sob) which at times developed into a scream. When tickled it retracted its lips, threw back its head, and the respirations became rapid and vocal. The condition just described was like that of a smile and the respiratory sound was like that of a whispered "Ha, Ha, Ha." When Brown stimulated the caudal pole of the optic thalamus he obtained respiratory changes similar to those when the intact animal was tickled (8). The stimulation of this part of the thalamus brought about *physical* reactions similar to those accompanying emotional states, but the author is careful to point out that "it cannot be assumed that a concomitant of the electrical stimulation is the conditioning of the emotion with which in every-day life we associate the reaction. It is more probable that in this stimulation we are only activating a link in the chain of mechanisms which normally take part in the state." Brown's other experiments (10) on the red nucleus are also of interest. Using the same, but decerebrated, chimpanzee he stimulated the region of the red nucleus, about 3 mm. above the anterior colliculi. The stimulation on one side produced usually homolateral flexion and contralateral extension, although at times a contralateral flexion was found. It would therefore appear that the red nucleus on one side has a bilateral connection, and that there is a compound or varying function (assuming that the spread of the current had been guarded against).

The contribution of van Valkenburg on the corpus callosum (52) is also of great interest. He gives a general review of about 50 articles dealing with callosal functions, results of personal experiments on the brains of rabbits, and accounts of 6 pathological cases in man. He disputes Dejerine's position that the occipital lobes are interconnected by way of the callosum, but does admit a fairly close connection of the frontal portions of the cerebrum. The precentral region on one side appears to be connected with the precentral and the postcentral regions on the opposite side. His general conclusions regarding the functions of the corpus callosum are negative. One is as follows: "The inability to follow the com-

missural connections of the frontal lobes, even though they probably exist, adds to our ignorance of the functional significance of these regions and renders physiological conclusions on this part of the corpus callosum impossible."

Sensory Functions.—As has already been indicated, Rothmann and Amantea hold to the view that the areas surrounding the central fissure of man, or of corresponding regions in animals, are sensory-motor. This is the view upheld by Luciani, and the experiments of Amantea (1) indicate how difficult it is to separate the sensory and motor functions. We have already considered this in relation to Southard's contribution (49). Amantea applied strychnine to the socalled motor cortex and obtained clonic movements upon stimulating these areas, which movements were not like those in an animal without strychnine. The motor phenomena were accompanied by what the author believes to be a hyperesthesic area of the skin, and he explains the motor differences in terms of sensory changes. It is just as easy, it seems no more improbable, to consider the motor difference in such cases to be directly motor rather than indirect phenomena. The matter can best, and can probably only, be settled by the results of careful examination of human pathological material, and such examinations indicate a separation of the motor area from the area concerned with sensory impulses from the skin and underlying tissues. This is indicated by the examinations of Sittig (48) and of Schröder (47). Although the results of their conclusions have not been confirmed anatomically by examination of the brains, the clinical facts warrant the belief that there were cortical lesions. Sittig described three cases, showing respectively paresthesia of the right thumb and the left angle of the mouth, a paresthesia between the thumb and index finger on the left hand and on the left upper part of the lip, and a paresthesia at the right angle of the mouth and on the right hand. Schröder's case showed a segmental distribution of the sensory disturbance limited to the two ulnar fingers and part of the middle finger, similar to the peripheral ulnar sensory "paralysis." There was hypalgesia, hyperesthesia, joint sensibility disturbance, but no difference in the two-point threshold and no temperature disturbance. This case was certainly due to cerebral accident. Kramer (30) also describes a case with subcortical lesion in which there were peculiar sensory disturbances of the nature of allochiria.

The sensory disturbances due to postcentral cortical lesions have been elevated to the dignity of a syndrome by Dejerine. That

author and Mouzon (14) have described two additional cases, and Roussy and Bertrand (46) have contributed one case. In general, it has been found that lesions of the postcentral regions bring about complete or almost complete alteration of tactile discrimination, a marked alteration of the sense of attitude and astereognosis, but with integrity of the senses of pain and temperature, and of bony sensibility. Spiller (50) says that "there can now be little doubt that the parietal lobe is the great cortical sensory area; it is possible that sensation is represented in its different qualities in different parts of this lobe, and it is probable that different segments of the body are separately represented as regards sensation in the parietal lobe, the sensory area being near the corresponding motor area." On the other hand he says that there is no evidence to warrant the belief that the sensory fibers conducting certain kinds of impulses are collected in special parts of the internal capsule.

Of the subcortical centers for the skin and underlying tissues the thalamus has been most frequently open to study. Roussy described a so-called thalamic syndrome, which has been disputed and confirmed by others. Holmes returns to the problem (23) and shows that matters are not as simple as they have been believed to be. The thalamic syndrome, due to lesions of the thalamus, consists in an hemianesthesia or hypesthesia for all forms of superficial sensibility, and relatively greater disturbances of deep sensibility, associated with spontaneous pains, but although there is no real paralysis there is often an ataxia or choreiform or athetoid movements. Holmes points out that the spontaneous pains are not constant in location, but that they shift from side to side, and often are replaced by a dull sensation like an uncomfortable constriction. The over-reaction which was supposed to be constant is not due to a hyperexcitability, because more intense stimuli are needed. Several types may be differentiated, Holmes believes. If the lower portion of the thalamus be affected we get high threshold values for all forms of sensation, but if the fillet and its terminations escape only those forms of sensation will be affected which are concerned with the cerebral cortex.

Bramwell has described a number of cases of occipital lobe lesions which were accompanied by visual disturbances (6, 7). The most interesting case was one in which central vision was diminished, and this was found to be due to a lesion of the calcarine area. He concludes that the calcarine core serves as a basis for macular as distinct from panoramic vision, and also concludes that macular

vision is "an evolution dependent upon (1) the development of binocular vision, *i. e.*, on the employment of corresponding points of the retinæ simultaneously, and on (2) the simultaneous development of the capacity to pay prolonged and individual attention to particular points of the general visual panorama. Macular vision is thus superimposed on the neuronic apparatus for panoramic, and the two types shade into one another." Dimmer has also described cases in which the central visual apparatus was injured (15). He believes that the macula has a representation in the calcarine fissure, and that the facts of his second case speak for the localization of this area at the posterior portion of the fissure. Pick's study (41) is not of special value in connection with localization, but gives interesting data regarding "orientation." Rothmann's cases (44) also tend to confirm the localization of the visual central apparatus in the occipital lobes. The condition in Rothmann's cases is more interesting in view of the recovery which took place.

The general facts obtained on brainless animals by Zeliony (56) and by Karplus and Kreidl (27) have already been discussed. The former extirpated the hemispheres of dogs and found that, although conditioned reflexes to light could not be obtained, light stimuli did bring about pupillary movements and at times the animals turned the head toward the source of the stimulation. There is no certainty that these reactions are more than reflex. Karplus and Kreidl also found with one hemisphere removed that there were no disturbances of the eye movements, except a temporary nystagmus in one animal, and there was an apparent restitution to such an extent that casual observation failed to show any defect. When both hemispheres had been removed the movements of the head and eyes did not seem to be affected, although two animals did show nystagmus, but the pupils reacted to light stimuli. Here also it is not possible to say that the reactions to light stimuli were at all like those which a normal animal would have.

Zeliony's observation regarding taste reactions is of interest (56). He noted that the dogs deprived of their cerebral hemispheres ate when meat was given to them and that meat containing quinine was rejected. Whether or not this is a reflex phenomenon could not be settled.

Speech and Other Association Functions.—A general review of the subject of aphasia has been written by Mingazzini (38). He holds neither to the newer view of Marie regarding the intimate relation of the lenticular nucleus to motor aphasia, nor to the older cortical

Broca's area view, for the destruction of either or both of these areas may be productive of an inability to speak. He enlarges the so-called Broca area to include the anterior portion of the insula and the operculum Rolandi, but he would not exclude the lenticular nucleus as an element in the prodcution of speech. In the cortical regions are to be found the hypothetical "engrams" or motor syllable images (in the motor speech areas). Sensory aphasia is due to lesions of the temporal areas, the transverse posterior temporal and the posterior third of the superior temporal. It is not possible, the author believes, to differentiate an area destruction of which brings about an amnesia for names, nor can verbal blindness or agraphia be said to be associated with defined lesions.

The work of the late Hughlings Jackson has been made the occasion of a special number of *Brain*, with an introductory article by the editor, Henry Head (**22**). Head points out that as early as 1868 Jackson showed that aphasic patients could be divided into two classes, those who could say nothing or little, and those who could speak but used words wrongly. The following quotations serve to give the main substance of the article: "The higher and voluntary aspects of speech tend to suffer more than the lower or more automatic, ... Writing is affected, not as a separate 'faculty,' but as a part of the failure to propositionize words. . . . External and internal speech are identical. . . . In the majority of cases of affections of speech mental images are unaffected. . . . (This) has been entirely neglected by neurologists. For almost every hypothesis propounded in the last forty years presupposes some defect in 'auditory' or 'visual word images'." It is encouraging to find at least one neurologist who doubts the accuracy of the assumption of the definite mental-cerebral relations, which assumption has been the reason for more unscientific work in this field than any other single thing.

In the discussion on motor aphasia, anarthria, and apraxia before the International Congress of Medicine at London the papers by Dejerine and by Liepmann were of the greatest value, giving views widely different but unfortunately crowded with the mental-cerebral assumptions which have been disputed by Head, and which are not supported by careful analyses of the material. Dejerine takes up briefly the history of motor aphasia (aphemia) and of anarthria, and distinguishes the two (**13**). By anarthria Dejerine means a difficulty in speaking which is of motor origin, such as spasm, paralysis, or incoördination. By motor aphasia he desig-

nates those disorders due to a loss of vocal representation of words, or which result from the loss of ideo-motor power. The true motor aphasia of Broca is, according to Dejerine, due to a loss of motor images of articulation. Anatomically, anarthria may be due to lesions of the medulla oblongata, or of the cerebral cortex, or of the cortico-bulbar fibers, and when lesions are bilateral a permanent anarthria may be produced by destructions of the opercula Rolandi, but the lenticular nucleus does not, the author believes, have a direct or intrinsic relation to the condition. In discussing those cases which are negative, with lesions of the left third frontal without motor aphasia, Dejerine assumes them to be due to the fact that the individuals were not really right-handed. He says they were cases of ambidexterity or of sinistrality, who have appeared to be right-handed on account of their education. These negative cases, which threatened to overturn the whole of the classical aphasia doctrines, are, therefore, not in opposition to but in accordance with the views expressed by Broca and by Wernicke, and the theory of aphasia remains intact.

Liepmann has discussed in more detail the subject of apraxia (**32**). He defines apraxia as the loss of the ability to execute movements for purposes. The acquirements of the motor apparatus can be considered in three ways, and disturbances of the ways in which the motor apparatus may work lead respectively to (1) ideational apraxia, (2) limb (*Glied*) apraxia, and (3) ideo-kinetic apraxia. This also applies to the motor speech organs. Thus, amnestic aphasia, or the difficulty in "finding" words, is an amnestic apraxia. Praxia, the normal condition, presupposes the use of the motor apparatus without an object, as for example the use of the arm and hand in making sawing movements when no saw is used. This is true for the vocal apparatus, of which at least part (the mouth) is intended to deal with food, but in speech there is a "free, objectiveless movement." "The inferiority of the right hemisphere for phasia and praxia can be referred to one and the same deficiency, *i. e.*, that of not directing movements without objects." The work of Liepmann is, like that of Dejerine, loaded with cerebral-mental assumptions, such as "memory for innervation complexes is one which has no equivalent in the consciousness" although "for simple and much practiced movements . . . such a pure kinetic memory" exists.

Tonic perseveration which was described by Wilson, and symptoms of which have been described in some apraxia cases by Liepmann, is the subject of a special communication by Wilson and

Walshe (55). They describe three cases with tonic perseveration, with differing concomitant symptoms. One patient had apraxia and hemiplegia, and after the removal of a large tumor from the right hemisphere the tonic innervation disappeared, leaving only a typical hemiplegia. A second patient had a tumor removed from the brain and the abnormal tonicity disappeared but a slight hemiplegia remained. The tonic innervation may occur without praxia disturbances, but it is usually associated with apraxia or dyspraxia, because of the location of the lesion in the frontal area.

The treatment of aphasia, the recovery of speech function by aphasics, has been the subject of a number of articles since Broca first expressed the belief that reëducation would be of advantage in these cases. This matter has recently been dealt with by Wilson and by Fröschels. Wilson (54) does not advocate the artificial methods recommended by most of the German writers, as illustrated by Gutzmann, but takes the natural method of teaching names rather than individual sounds. He concludes that there is evidence to warrant the belief that auxiliary mechanisms are developed in undamaged areas of the cerebrum. Fröschels (20, 21) cites results in seven cases, but gives no adequate account of the course of the reëducation. He has used the method of saying a word in the patient's ear, and then showing him the mouth as it is placed to say the word. Some patients may learn to read the lips, as do the deaf. In his second paper he advocates the early training of these defectives, a view consistent with what we know of other cerebral defectives.

On the negative side Dupuy (16) cites facts which he considers do not warrant the localizations which have been made. He ridicules the explanations of Mendel's case, aphasia with rightsided lesion, that the patient was not really righthanded, for he points out that the family of the patient were righthanded and that if the patient had been normally lefthanded such a condition would, or should, have been evidenced in the family at some point. He believes that most, if not all, of the facts can be understood on the Brown-Séquard hypothesis of inhibition. Bickel (3) has also dealt with a negative case of lesion of Broca's area and of the first temporal lobe on the left in a righthanded individual. This patient gave no prominent signs of motor aphasia, although there was a slowing and an unclearness of speech, and some difficulty in finding words.

Bianchi (2) has dealt with the relations of the frontal lobes, and

has shown that they have connections with all other parts of the cerebral cortex, but with none of the basal ganglia except the putamen. "The most certain statement that we can make is that the frontal lobe has direct relations with the motor zone by means of the short fibers, and direct relations with the other parts of the cortex through two great bundles." Lesions of this region do not produce disturbances of psychical activities. By a comparison of the clinical findings and the extent and severity of frontal lobe atrophies Franz (**18**, part 1) has come to the conclusion that there exists no direct relation.

References

1. Amantea, G. Sur les rapports entre les centres corticaux de la circonvolution sigmoïde et la sensibilité cutanée chez le chien. *Arch. ital. de biol.*, 1915, **63**, 143–148.
2. Bianchi, L. Recent Researches on the Functions and Anatomical Relations of the Frontal Lobes. *Trans. XVII Intern. Cong. of Med.*, 1914, Sect. XII, Part 2, 17–24.
3. Bickel, H. Zur Kasuistik der klinisch negativen Fälle von Aphasie. *Neur. Centbl.*, 1914, 33, 287–291.
4. Bikeles, G. & Zbyszewski, L. Ueber Erregbarkeit der Grosshirnrinde und Auslösbarkeit von Rindenepilepsie unter Einfluss von Schlafmitteln wie nach Verabreichung grösserer Bromgaben. *Pflüger's Arch. f. d. ges. Physiol.*, 1914, **158**, 235–251.
5. Boeke, J. Die Regenerationserscheinungen bei der Verheilung von motorischen und rezeptorischen Nervenfasern. *Pflüger's Arch. f. d. ges. Physiol.*, 1914, **158**, 84–91.
6. Bramwell, B. Bilateral Lesion in the Occipital Lobes Correctly Diagnosed Twenty-four Years before Death, with Histopathological Report by Dr. Shaw Bolton, Showing the Portion of the Cortical Area for Macular as Distinct from Non-macular or Panoramic Vision. *Edinb. Med. J.*, 1915, **15**, 4–18.
7. Bramwell, B. Lesions of the Occipital Lobe and Affections of Vision. *Edinb. Med. J.*, 1915, **15**, 165–185.
8. Brown, T. G. Note on the Physiology of the Basal Ganglia and Mid-brain of the Anthropoid Ape, Especially in Reference to the Act of Laughter. *J. of Physiol.*, 1915, **49**, 195–207.
9. Brown, T. G. On the Activities of the Central Nervous System of the Unborn Foetus of the Cat; with a Discussion of the Question Whether Progression (Walking, etc.) is a "Learnt" Complex. *J. of Physiol.*, 1915, **49**, 208–215.
10. Brown, T. G. On the Effect of the Artificial Stimulation of the Red Nucleus in the Anthropoid Ape. *J. of Physiol.*, 1915, **49**, 185–194.
11. Brown, T. G. On the Phenomenon of Facilitation. 1. Its Occurrence in Reactions Induced by Stimulation of the "Motor" Cortex of the Cerebrum in Monkeys. *Quart. J. of Exper. Physiol.*, 1915, **9**, 81–99.
12. Claude, H., Dumas, R., & Porak, R. Adaptation fonctionnelle par suppléance dans les paralysies traumatiques des nerfs. *Presse méd.*, 1915, **23**, 205–207.
13. Dejerine, J. Motor Aphasia, Anarthria, and Apraxia. *Trans. XVII Intern. Cong. of Med.*, 1914, Sect. XI, Part 1, 85–106.

14. DEJERINE, J. & MOUZON, J. Deux cas de syndrome sensitif cortical. *Rev. neur.*, 1914, **22**, 388–392.

15. DIMMER, F. Zwei Fälle von Schussverletzungen des zentralen Sehbahnen. *Wien. klin. Woch.*, 1915, **28**, 519–524.

16. DUPUY, E. On Localization of Motor and Speech Centers in Definite Areas of the Cortex of the Brain. *Lancet*, 1914, **187**, 207–210.

17. FÖRSTER, O. Relations between Spasticity and Paralysis in Spastic Paralysis. *Trans. XVII Intern. Cong. of Med.*, 1914, Sect. XI, Part 2, 55–64.

18. FRANZ, S. I. On the Functions of the Cerebrum: (1) Symptomatological Differences Associated with Similar Cerebral Lesions in the Insane; (2) The Variations in Distribution of the Motor Centers. *Psychol. Monog.*, 1915, **19**, No. 81. Pp. 162.

19. FRANZ, S. I., SCHEETZ, M. E. & WILSON, A. A. The Possibility of Recovery of Motor Function in Long-Standing Hemiplegia. *J. of Amer. Med. Assoc.*, 1915, **65**, 2150–2154.

20. FRÖSCHELS, E. Ueber die Behandlung der Aphasien. *Arch. f. Psychiat. u. Nervenkr.*, 1914, **53**, 221–261.

21. FRÖSCHELS, E. Uebungsschulen für Gehirnkrüppel. *Münch. med. Woch.*, 1915, **62**, 913.

22. HEAD, H. Hughlings Jackson on Aphasia and Kindred Affections of Speech. *Brain*, 1915, **38**, 1–27. (Pp. 28–190 of the same number are reprints of some of Jackson's papers and a bibliography of 36 titles.)

23. HOLMES, G. The Symptoms and Pathology of Diseases of the Optic Thalamus. *Trans. XVII Intern. Cong. of Med.*, 1914, Sect. XI, Part 2, 65–73.

24. JEFFERSON, G. Cortical Localization and Furrow Formation. *J. of Comp. Neur.*, 1915, **25**, 291–299.

25. KARPLUS, J. P. Experimenteller Beitrag zur Kenntnis der Gehirnvorgänge beim epileptischen Anfall. *Wien. klin. Woch.*, 1914, **27**, 645–651.

26. KARPLUS, J. Ueber Hemisphärenexstirpationen bei Hapale und Macacus. *Jahrb. f. Psychiat. u. Neur.*, 1914, **36**, 243–256.

27. KARPLUS, J. P. & KREIDL, A. Ueber Totalexstirpationen einer und beider Grosshirnhemisphären an Affen (Macacus rhesus). *Arch. f. Anat. u. Physiol.* (Physiol. Abt.), 1914, 155–212.

28. KENNEDY, R. Experiments on the Restoration of Paralyzed Muscles by Means of Nerve Anastomosis. *Phil. Trans.*, 1914, **B. 205**, 27–76.

29. KOUINDJY, P. La kinésitherapie dans le traitment des maladies nerveuses. *Trans. XVII Intern. Cong. of Med.*, 1914, Sect. XI, Part 2, 33–41.

30. KRAMER, —. Alloästhesie und fehlender Wahrnehmung der gelähmte Körperhälfte bei subcorticalem Hirnherd. *Berl. klin. Woch.*, 1915, **52**, 301–302.

31. KRASNOGORSKI, N. On the Fundamental Mechanisms of the Activity of the Cerebral Cortex in Children. *Trans. XVII Intern. Cong. of Med.*, 1914, Sect. X, Part 2, 199–200.

32. LIEPMANN, —. Motor Aphasia, Anarthria, and Apraxia. *Trans. XVII Intern. Cong. of Med.*, 1914, Sect. XI, Part 2, 97–106.

33. LLOYD, J. H. The Morphology and Functions of the Corpus Striatum. *J. of Nerv. & Ment. Dis.*, 1915, **42**, 370–382.

34. MARBURG, O. & RANZI, E. Erfahrungen über die Behandlung von Hirnschüssen. *Wien. klin. Woch.*, 1914, **27**, 1471–1473.

35. MAYER, L. Die orthopädische Behandlung der alten Hemiplegiker. *Berl. klin. Woch.*, 1915, **52**, 606–607.

36. Mills, C. K. Concerning Cerebral Morphology in its Relations to Cerebral Localization. *J. of Nerv. and Ment. Dis.*, 1915, **42**, 322, 357.
37. Mills, C. K. Muscle Tonicity, Emotional Expression, and the Cerebral Tonetic Apparatus. *Neur. Centbl.*, 1914, **33**, 1266–1280.
38. Mingazzini, G. Ueber den gegenwärtigen Stand unserer Kenntnis der Aphasielehre. *Monat. f. Psychiat. u. Neur.*, 1915, **37**, 150–197.
39. Misch, W. & Lotz, A. Muskelaktionsströme bei organischen und funktionellen Erkrankungen des Zentralnervensystems. *Monat. f. Psychiat. u. Neur.*, 1914, **36**, 191–196.
40. Nammack, C. E. Progressive Lenticular Degeneration. *Med. Rec.*, 1914, **86**, 997–1000.
41. Pick, A. Zur Lokalisation in den Sehbahnen, mit einem Beitrage zur Lehre von den Störungen der Orientierung im Raum. *Prag. med. Woch.*, 1915, **40**, 81–82.
42. Poppelreuter, W. Ueber psychische Ausfallserscheinungen nach Hirnverletzung. *Münch. med. Woch.*, 1915, **62**, 489–491.
43. Robinson, R. Les localisations physiologiques de l'encéphale en contraste avec les destructions étendues de cet organ. *C. r. acad. d. sci.*, 1913, **157**, 1463–1464.
44. Rothmann, M. Die Hirnphysiologie im Dienste des Krieges. *Berl. klin. Woch.*, 1915, **52**, 338–341.
45. Rothmann, M. Ueber die Grenzen der Extremitätenregion der Grosshirnrinde. *Monat. f. Psychiat. u. Neur.*, 1914, **36**, 319–341.
46. Roussy, G. & Bertrand, J. Un cas de Syndrome Sensitif Cortical par blessures de guerre. *Rev. neur.*, 1914, **22**, 396–398.
47. Schroder, —. Segmentartige kortikale Sensibilitätsstörungen von ulnaren Typus. *Münch. med. Woch.*, 1915, **43**, 623.
48. Sittig, O. Klinische Beiträge zur Lehre von der Lokalisation der sensiblen Rindenzentren. *Prag. med. Woch.*, 1914, **39**, 548–550.
49. Southard, E. E. The Association of Various Hyperkinetic Symptoms with Partial Lesions of the Optic Thalamus. *J. of Nerv. & Ment. Dis.*, 1914, **41**, 617–640.
50. Spiller, W. Remarks on the Central Representation of Sensation. *J. of Nerv. & Ment. Dis.*, 1915, **42**, 399–418.
51. Trendelenburg, W. Untersuchungen über den Ausgleich der Bewegungsstörungen nach Rindenausschaltungen am Affengehirn. *Zsch. f. Biol.*, 1914, **65**, 103–138.
52. Valkenburg, C. T. van. Experimental and Pathologico-Anatomical Researches on the Corpus Callosum. *Brain*, 1913, **36**, 119–165.
53. Welty, C. F. Anastomosis of the Facial and Hypoglossal Nerves for Facial Paralysis. *J. of Amer. Med. Assoc.*, 1915, **62**, 612–614.
54. Wilson, S. A. K. Aphasia and its Treatment. *Practitioner*, 1915, **94**, 683–699.
55. Wilson, S. A. K. & Walshe, F. M. R. The Phenomenon of "Tonic Innervation" and its Relation to Motor Apraxia. *Brain*, 1915, **37**, 199–246.
56. Zeliony, G. P. Observations sur des chiens auxquels on a enlevé les hémisphères cérébraux. *C. r. soc. de biol.*, 1913, **74**, 707–708.

REFLEX MECHANISMS AND THE PHYSIOLOGY OF NERVE[1]

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The most important general discussion of the past two years is probably to be found in the two papers by R. S. Lillie (**41**, **42**) on the nature of excitation and conduction especially in nerve tissue. Arguing in part from the close positive correlation which is found in many muscle and nerve tissues "between the rate at which the electrical variation [action-current] rises to its maximum at any region of the excited tissue, and the rate at which the excitation-wave is propagated in that tissue," Lillie (**41**) comes to the view "that the electrical variation is itself the essential feature of the excitation-process, and constitutes that functional component of the local process which directly excites the adjoining regions of the tissue to activity; if so, its rate of development must determine the rate at which excitation is transmitted from the active region of the tissue to those adjoining." The author suggests that the action-current initiated by local stimulation itself stimulates electrically the nerve *at a distance* from the locus of excitation—a distance, it may be, of 2 or 3 centimeters. The "rate at which" the "electrical variation rises to its maximum is the chief factor in determining the velocity of conduction"; but a second factor is the maximal distance from the already active region at which the action-current there will cause a second stimulation (action-current) farther on along the nerve. This would seem to make conduction proceed by discrete leaps. Why the action-current at the point of stimulation should not first excite adjacent points, and these the next-adjacent, is not made clear: yet if conduction *is* thus continuous, rather than discrete, the second factor cited as governing the velocity of conduction would not figure (cf. p. 437). Several interesting facts are adduced to show that the electrical variation is not merely an index of some underlying physiological process, but is itself the central fact. In living tissues in general the rate of conduction "shows no appreciable correlation with ionic or other diffusion-velocities, nor with the transmission-velocities of mechanical, thermal, or electrical influences." In the second paper (**42**) Lillie

[1] For previous reviews of these topics see PSYCHOL. BULL., 1911, **8**, 119–130, 136–139; 1913, **10**, 146–152; 1914, **11**, 140–150.

describes a modified form of Bernstein's "membrane theory" of the bio-electric processes. The nerve is surrounded by a semipermeable membrane. Ordinarily this is probably permeable to certain cations only, but stimulation renders it (at the point stimulated) permeable to ions of both classes. Thus the potential difference between the interior of the nerve and the outer medium is suddenly lowered at the site of stimulation. "During the resting condition cations are unable to leave the cell because of the impermeability of the surface-film to anions and the resulting electrostatic stress; the normal polarized condition of the resting element (with outer surface positive) is thus accounted for. If then at any point in the surface the membrane is altered by stimulation so as to allow anions to leave the cell, the electrostatic tension holding back cations is at once released—not only at the region of stimulation, but at all other points of the cell-surface; cations are then free to leave the cell in numbers equivalent to the anions; in other words, a current then flows between unaltered and altered regions. . . ." Within the cell this current flows from active towards inactive regions, and in the external medium in the reverse direction. Nerve conduction involves the passage of a permeability- or depolarization-wave. Both papers are admirably clear. Lillie has elsewhere (43) printed a more popular exposition of the process of stimulation in living tissue.

McClendon (44) argues that "if stimulation consists in increase in permeability, we should expect anæsthetics to prevent this change." Working with eggs of the pike, he finds this to be the case. "Stimulation and anæsthesia seem to be antagonistic states." Similarly Osterhout, Joel (38), and others have found that anesthetics decrease permeability. Traube too (58, 59) finds this, but he believes that the result is indirectly attained. Owing to certain striking properties which are common to all narcotics, they readily penetrate the nerve-cell walls, and then dissolve or, better, precipitate the lipoids contained inside. The precipitates accumulate on the inner surface of the cell-membrane, and thus create a "dead space" which renders the membrane impermeable. The cell cannot now be stimulated. While the narcotic is penetrating the cell, and before precipitates have formed, the tissue presents a phase of increased excitability. Traube criticizes the theory of Höber and Joel (38) according to which the tissue should exhibit a phase of depression followed, in the deeper stage of anesthesia, by exaltation: which would not agree with common observation.

While Lillie makes electrical phenomena the central factor in the processes of stimulation and conduction, he does not seek to deny certain other phenomena which would seem to relate these processes more closely to "metabolism" in the familiar sense (**43**, p. 589). But other observers emphasize the metabolic aspects more strongly. Thus Montuori (**49**) argues for Verworn's well-known theory that the essence of narcosis (or anæsthesia) is asphyxiation. He finds that by gradually raising the temperature (from 18° C. to 28° C.) of the water in which tadpoles of *Bufo vulgaris* are living, he has rendered them more resistant to both asphyxiation and narcotics (alcohol, ether, chloroform, etc.). Tashiro and Adams (**56**) believe that "oxygen is a primary factor in the excitability of the nerve, as well as in the conductance of the nerve impulse"; and that protoplasmic irritability is essentially a chemical process. Gottschalk (**32**) holds that the nerves of cold-blooded animals contain some reserve of oxygen, "probably in loose chemical combination"; and "a smaller quantity in physical solution." And Thörner (**57**) shows that the irritability of medullated nerve (frog) is higher in oxygen than in air (though its conductivity is not modified). Moldovan and Weinfurter (**48**) believe, because the narcotics fall into *one* serial arrangement with regard to several distinct chemical properties (power to dissolve lipoids, to asphyxiate, to destroy ferments, etc.), that the fundamental nature of narcosis is probably different in different tissues. They believe that in the "cerebral cortex" narcosis is primarily suffocation; but that in "nerve tissue" (axone?) it is something else. Tashiro and Adams (**56**), from measurements with the biometer on the claw nerve of the Spider Crab, conclude that "there is a perfect parallelism between the state of excitability and the CO_2 output." "All irritable tissues give off CO_2, resting nerves being no exception": and "when irritable tissue is stimulated, this CO_2 production is increased"; it is greatly depressed by anæsthetization. They suggest that there are two metabolic phases in nerve: (1) The formation of an organic peroxide; (2) the "decomposition of this peroxide by peroxidase" (as held by Gasser and Lovenhart). Sanders (**53**) finds that local warming produces a region of decrement in the frog nerve: if the nerve is plentifully supplied with oxygen, more heat is required to produce this decrement. Benda (**5**) insists strongly on the difference between the irritability of a nerve and the efficiency of it after it is stimulated ("*Leistungsfähigkeit*").

The all-or-none law seems to be meeting with wide acceptance.

Adrian (1), after an interesting discussion, says: "We may conclude that there is an all-or-none relation between the stimulus and the propagated disturbance [in nerve] because the disturbance will always be set up as soon as the local excitatory change has reached a certain critical intensity at any point on the nerve; any further increase in the local change will have no effect on the propagated disturbance on account of the refractory state which is developed as soon as the disturbance is set in motion." In agreement with this last point Eichholtz (23) finds that the *minimal* summation-time of a spinal reflex (frog) is "dependent on the refractory period of the afferent nerve." Adrian (3) finds that change of temperature affects all phases of the recovery period alike, so that probably a single process accounts for all the phenomena of the refractory state. Adrian, further (2), confirms the observation of Koike (disputed by Fröhlich) that the rate of conduction in a region of decrement is constant, not becoming slower as the impulse becomes feebler. This rate is, however, everywhere slower than in normal nerve. Rehorn (52) states that in a region of decrement the decrease of the nervous impulse is a "linear function" of the distance traversed (but for the validity of this cf. Rehorn, p. 65). In asphyxiation the propagated disturbance suffers earlier than the local excitatory process. Fredericq (30) finds that under increasing mechanical compression conduction remains normal until at some point conduction suddenly ceases entirely. If the compression is removed conduction as suddenly returns. Hoffmann (35) offers evidence against the all-or-none law holding for the nerves of crustaceans (crab and lobster). He does not seek to argue against the principle in the case of vertebrates. But Uexküll and Tirala (61) raise what is probably a valid objection to Hoffmann's interpretation of his work with crustaceans. They also offer the view that the "inhibitory" crustacean nerve functions by "sucking" nervous energy out of the muscle. Elrington (24) thinks that the lowered reflex threshold (frog) in strychnine poisoning is incompatible with the all-or-none law: but since "with faradic stimulation it can be shown that the weakest stimuli which are able under strychnine poisoning to produce a reflex contraction, have always also an effect in the normal spinal cord," he admits that the all-or-none law is still valid. The reviewer does not at all follow the author's reasoning. Forbes and Gregg (29) express themselves with considerable reserve regarding the validity of the all-or-none principle. By means of a special arrangement of electrodes laid on an arthropod nerve Hoff-

mann (**34**) has observed the way in which two nerve impulses travelling in opposite directions on the same fiber pass each other. Impulses produced by induction shocks at 50 per sec. if maximal interfere with and cancel each other: if submaximal they weaken but succeed in passing each other, and show something approaching an algebraic summation of their intensities. Hoffmann accepts the all-or-none principle, in vertebrates, for both nerve and muscle. By others the law is held to be more securely established for nerve than for muscle.

Boeke (**8**) has continued his interesting experiments in regeneration. Regenerating fibers, whether sensory or motor, once started to grow along a path formerly occupied by a (now degenerated) fiber, "cannot leave" this path. Thus Boeke succeeded in making a chiefly sensory nerve (lingualis, of the hedgehog) regenerate along the path formerly occupied by a chiefly motor nerve (hypoglossus). This nerve finally "formed terminal arborizations on the muscle-fibers" which at least so far restored the motor innervation of the muscle as to stop the fibrillary tremor exhibited by the muscle after its proper motor nerve had been cut, and also to save the muscle from degeneration. Likewise motor fibers (hypoglossus) having regenerated in the path of the sensory lingualis either restored or in some way caused to reappear the degenerated taste-buds.

Troland (**60**) presents an interesting view of sensory adaptation as a chemical equilibrium. The theory appears to coincide with empirical facts better than does the well-known theory of Hering, and it avoids Hering's very dubious assumption that stimuli excite anabolic as well as katabolic processes in protoplasm. Troland succeeds in lucidly stating physiological fact in mathematical notation. The paper is important in several respects.

Schwenker (**54**) finds three kinds of contraction process in striated muscle: the first is rapid and rapidly reversible; the second is slow and less rapidly reversible; the third is slow, irreversible, and connected with the death of the muscle. The second sort is probably responsible for general muscle tonus. Now in "tonic innervation" Ernst (**25**) finds that no carbohydrates are consumed. He believes that slow muscle tonus and rapid muscle jerk depend on different chemical processes going on in different substances. Jensen (**37**) dissents from this two-substance theory; but he believes that tonic contraction is maintained without flow or "mobilization of electrolytes," while the rapid jerk involves such mobilization. From his work on the thermal stimulation of muscle, he concludes

that all contraction is based on the katabolism of an unstable substance which, once initiated, continues of itself. Heat, he believes, stimulates muscle directly, and not through the intermediation of "autoelectric stimuli." Mansfeld and Lukács (**45**) show that striated muscles have two sorts of motor end-organs and two forms of innervation; by medullated and by non-medullated (sympathetic) fibers. Tonic innervation, which they call "chemical tonus" (and of which Mansfeld (**46**) states that the "respiratory metabolism" is the result and index), is maintained even in skeletal muscle *solely* by the sympathetic fibers. This appears to be an important paper. On the other hand, Kuno (**39**) contends that at least in *Rana esculenta* the sympathetic system plays no part in maintaining the tonus of the hind limbs. He finds the anterior roots of the cord, however, to be important here. Weizsäcker (**62**), from myothermic experiments on the rapid muscle jerk, concludes that this involves "two different machines"—(1) a contraction mechanism and (2) an oxidative recovery mechanism. The first is non-oxidative and consists in turn of two part-processes, "a part which provides energy, and a part which transforms this into mechanical energy: the former can be active without the latter" (in which case the energy provided turns into heat). This energy yielded by the first "part" comes from some "non-oxidative" store, as say, of glycogen or sugar. Meigs (**47**) objects to an earlier paper of Hürthle who had undertaken to show that striated muscle on contracting does *not* increase in volume. Meigs supports Engelmann's "*Quellungshypothese*." Hürthle (**36**) replies that his former findings against this hypothesis are valid. Snyder (**55**) holds that smooth muscle contracts and maintains its contraction with less expenditure through oxidation than does striated muscle. From indirect stimulation of smooth muscle he records an "absence of any measurable amounts of heat production accompanying development of tension." The gaseous exchanges in cat muscle under decerebrate rigidity are notoriously low. Langley (**40**) finds "that curari and nicotine produce their effects by an action on the neural region of the muscle fiber and not by an action on the nerve endings, so that the explanation of the specific action must depend upon a difference in the neural and non-neural region of the muscle fibers."

In connection with reflex process there has been considerable discussion as to the periodicity of the nervous impulse. In what will be one of his last published papers (**50**) the late Hans Piper refutes certain objections of Garten and Dittler, and reasserts his

earlier statement that the normal action-current of voluntary forearm flexion oscillates approximately 50 times per second. The fundamental unit of neuro-muscular action is the two-phase action-wave. In some persons ("Type II") the action-current of forearm flexion has a period much higher than 50 per sec., and inconstant. Piper believes that in such cases the action-currents of the separate fibrillæ are passing down the nerve out of phase with one another ("platoon fire"), so that the galvanometer records a varying and much higher number of oscillations. Even here the period in the individual fibrilla is probably 50 per sec. There is good evidence for this interpretation. In "Type I" the action-currents pass along the separate fibrillæ all in phase ("volley fire"), and the 50-rhythm is exhibited clearly. Any person appears to be permanently of one type or of the other; and Piper does not deem one more normal or advantageous than the other. There are persons of intermediate type. In extreme fatigue the 50-frequency goes down to 25 or even 20 per sec., although the amplitude of each action-wave remains the same. The paper is clear and of fundamental importance. It is confirmed in all points by Fahrenkamp (**26**) from his studies of the action-current in strychnine tetanus. The author inclines to the view that the 50-rhythm originates in the spinal cord. Gregor and Schilder (**33**) record the same 50-rate in continued voluntary contractions: in voluntary relaxation the rate decreases gradually to the point of complete relaxation (which directly confirms a point made by Forbes and Gregg (**29**): see below). The mental state of fear produces no characteristic change of rate; but when the rate has been decreased by fatigue (cf. Piper) a sudden fright increases the height of each crest, and still does not change the rate. A single "psychic impulse" to move the finger gives an action-current of at least 3 or 4 crests.

Dittler and Günther (**22**) dispute Piper's 50-rhythm. They find the normal action-current for voluntary innervation (man) to range from 120 to 240 oscillations per sec., with a general average at about 180 per sec. Beritoff (**6**), who does not accept the all-or-none principle for either nerve or muscle, has studied the action-currents of M. semitendinosus (frog) induced by faradic stimulation of the central stump of a severed posterior root. He finds the action-current fluctuating from 50 to 150 oscillations per sec., with a general average around 70 to 80 per sec. These are practically the same rates which the author found in previous experiments in which the posterior roots had not been cut, *i. e.*, in which proprio-

ception remained. The rhythm of the action-current of the muscle (and motor-nerve?) in the spinal reflex is largely, but not wholly, independent of the rate of faradic afferent stimulation. There is an after-discharge in which both frequency and intensity of the action-current gradually decrease. If the rate of stimulation is very slow it can generally be observed that the muscles receive 3 or 4 innervations, of decreasing strength, for each stimulation. This number is increased when the cordal segment involved is poisoned with strychnine. In three other connected papers (7) Beritoff records very similar results. With afferent stimulations of 20 to 40 per sec. the muscular action-current generally shows a group of 3 or 4 decreasing crests for each stimulation. If the stimulation frequency is 50 to 100 per sec. the muscular action-current is apt to show the very same frequency. It is to be hoped that a further understanding of "volley and platoon fire" will serve to bring these results into somewhat better agreement with those of other observers. Beritoff believes in a specifically "inhibitory" nerve-impulse, which "sums algebraically" with excitatory impulses. Hoffmann and Snyder (55) agree that in the knee-jerk, at least, "a skeletal muscle is made to contract by a normal reflex consisting of one, and one nervous impulse only," as contrasted with "a cataract of nervous impulses . . . poured in." Most authors, however, seem to accept the view of Ewald (1911), that reflex innervation, whether natural or artificial, is periodic innervation. Hoffmann (34) believes that for every muscle there is probably some optimal rhythm of excitation (cf. Sherrington).

Forbes and Gregg (29) have an important paper on the physiology of nerve and muscle, the technique of registering action-currents, and especially on the correlation between the strength of a reflex stimulus and the strength of the resulting muscular response. "It is a salient fact in connection with excitable tissues that they may respond to continuous stimulation discontinuously, *i. e.*, with a succession of separate propagated disturbances"; and this is of course especially true of the reflex arc. Now the all-or-none law of nerve-action raises a serious problem as to how the observed correlation between the strength of a reflex stimulus and that of the muscular response is produced. Reasons are given why this correlation "cannot be explained by the assumption that an additional afferent nerve fiber is excited at each perceptible increase in intensity of sensory stimulation." They suggest that the intenser stimuli produce a nerve impulse of higher *frequency* (each pulsation

being still "all-or-none"), which will in turn cause the muscle to contract more strongly. This seems to the reviewer a very important theory (for which, however, priority may perhaps be claimed by Verworn or his school). "The work of Lucas and Adrian shows that the recovery of excitability in nerve following its refractory period is such that a sustained stimulus of great intensity would produce a more rapidly rhythmic response than one of less intensity." Ewald showed in 1911 (compare **35**) that in crustaceans the strength of the contraction of the claw muscle depends on the frequency of the innervating impulse: and an observation by Gregor and Schilder (cited above) confirms the same view for human reflexes. It is true that Dittler and Günther (**22**) contend that the strength of voluntary contraction depends not on the frequency but on the intensity of the action-current. Forbes and Gregg (**28**) have also described an interesting recording camera for use in connection with the Einthoven galvanometer. They show that "stimuli occurring within less than one-hundredth of a second of each other appear to cause summation in the flexion reflex, while stimuli a tenth of a second apart fail to do so, the second response showing a decrement in consequence of the first."

Brown (**9**) studies rhythmic reflex movements of the "progression" type. "Rhythmic progression is conditioned by an equal balance of two antagonistic central activities," but it is remarkably independent of proprio-ceptive or other afferent mechanisms, and even it "may be exhibited in the lumbar spinal centers alone, and is then not conditioned by the activity of higher centers." "The rhythm may best be explained as conditioned by some function of the interconnexion of efferent neurones." The spinal center is compared to the respiratory center. This theory "tends to displace the reflex as the unit of activity in the spinal centers and to put in its stead a mechanism composed of efferent neurones. Perhaps the simplest unitary mechanism is one formed of the efferent neurones to two antagonistic muscles"; perhaps each such neurone, or "half-center," sends a side fiber to the other half-center, this fiber having the function of depressing that half-center. The efferent half-centers were probably once functionally independent of any afferent neurone. Brown's other papers (**10** to **21**, inclusive) deal with the compounding of reflexes in several sorts of spinal preparation. Important, and in some respects interesting, as these papers undoubtedly are, they present a vast mass of minute detail which is insusceptible of being reviewed, and a minimum of new

insight into the spinal mechanism. The reviewer was unable to discover any general *principles* here emerging which have not been described in previous papers by Sherrington. From stimulation of the motor cortex of the monkey it was observed (**18**) that the maximum time within which facilitation (summation) of liminal stimuli occurs, is about 10 seconds.

Asayama (**4**) studies the proprio-ceptive reflex as obtained by mechanically stretching a flexor muscle. The "stretch-reflex" can be evoked by an elongation (if it is not too slow) of one one-hundredth of the length of the muscle; the response is sometimes multiple (2–4 twitches); its latent period is less than that of "reflexes evoked from the muscle by other afferent channels." When all such reflexes are completely fatigued, the proprio-ceptive reflex "persists unaffected by that fatigue"; and this argues that the seat of reflex fatigue "lies mainly in some part of the reflex-arc on the afferent side of the motoneurones." Similarly Benda (**5**) finds that the depressing effect of Ca and Mg ions (introduced in the blood) seems to lie in the nerve (or its end-organ) rather than in the muscle, for it is hardly evident in direct stimulation of the muscle.

Porter (**51**) states that the flexion reflex is one whose threshold is normally low, and this is not greatly reduced by strychnine. But strychnine reduces the threshold of those reflexes whose threshold is normally high, to about that of the flexion reflex. In other words the proprio-lateral flexion reflex is normally often "in the highest state of irritability of which the mechanism is capable." Mansfeld (**46**) believes that very large doses of strychnine may lame the motor-endings of sympathetic nerves. Gildemeister (**31**) attributes the psycho-galvanic reflex mainly to activity of the sweat glands. He doubts whether the phenomenon should be called "psycho"-galvanic.

Forbes and Sherrington (**27**) find that "certain acoustic stimuli excite movements of the pinna, neck, tail and limbs in the cat even within a short time after complete removal of the cerebral hemispheres, striata, thalami, corpora geniculata," etc. "The turning of the head and perhaps the movements of the pinna appear interpretable as orientation reflexes, *i. e.*, reactions adjusting the organism in its relation to the spatial locus of the stimuli, and were the rest of the brain present, helping the organism to locate the sound." Some of the movements elicited by the sound stimuli were "mimetic," *e. g.*, similar to those that normally indicate anger.

Such sounds as "the barking of a dog, yowling of a cat, or the whistling of birds" were specially efficient in eliciting this mimesis.

References

1. Adrian, E. D. The All-or-None Principle in Nerve. *J. of Physiol.*, 1914, **47**, 460–474.
2. Adrian, E. D. The Relation between the Size of the Propagated Disturbance and the Rate of Conduction in Nerve. *J. of Physiol.*, 1914, **48**, 53–72.
3. Adrian, E. D. The Temperature Coefficient of the Refractory Period in Nerve. *J. of Physiol.*, 1914, **48**, 453–464.
4. Asayama, C. The Proprioceptive Reflex of a Flexor Muscle. *Quart. J. of Exper. Physiol.*, 1915, **9**, 265–279.
5. Benda, R. Ueber den Einfluss des Traubenzuckers, der Natrium-, Kalium- und Magnesium-Ionen auf die Reizbarkeit, Leistungsfähigkeit und Ermüdbarkeit des motorischen Nerven und des Skelettmuskels. *Zsch. f. Biol.*, 1914, **63**, 11–77.
6. Beritoff, J. S. Ueber die Erregungsrhythmik der Skelettmuskeln bei der reflektorischen Innervation. *Zsch. f. Biol.*, 1914, **64**, 161–174.
7. Beritoff, J. S. Die zentrale reziproke Hemmung auf Grund der elektrischen Erscheinungen am Muskel. I. *Zsch. f. Biol.*, 1914, **64**, 175–187; II, 289–300; III, 301–314.
8. Boeke, J. Die Regenerationserscheinungen bei der Verheilung von motorischen und rezeptorischen Nervenfasern. *Pflüger's Arch. f. d. ges. Physiol.*, 1914, **158**, 84–91.
9. Brown, T. G. On the Nature of the Fundamental Activity of the Nervous Centers; together with an Analysis of the Conditioning of Rhythmic Activity in Progression, and a Theory of the Evolution of Function in the Nervous System. *J. of Physiol.*, 1914, **48**, 18–46.
10. Brown, T. G. Immediate and Successive Effects of Compound Stimulation in Spinal Preparations. *Quart. J. of Exper. Physiol.*, 1914, **7**, 197–243.
11. Brown, T. G. Immediate Reflex Phenomena Resultant upon Compound Stimulation in Decerebrate Preparations. *Quart. J. of Exper. Physiol.*, 1914, **7**, 245–291.
12. Brown, T. G. Decerebrate Preparations, Successive Phenomena in Compound Reactions—Stimuli of Synchronous Termination and Stimuli of Asynchronous Termination where the Extension-Reflex is Left in Action. *Quart. J. of Exper. Physiol.*, 1914, **7**, 293–344.
13. Brown, T. G. Decerebrate Preparations, Successive Phenomena in Compound Reactions—Stimuli of Asynchronous Termination where the Flexion-Reflex is Left in Action. *Quart. J. of Exper. Physiol.*, 1914, **7**, 345–382.
14. Brown, T. G. The Successive Effects of the Compounding of Reflexes where the 'Pure' Reactions are Abnormal (Ipsilateral Extension or Contralateral Flexion) in Decerebrate Preparations. *Quart. J. of Exper. Physiol.*, 1914, **7**, 383–405.
15. Brown, T. G. The Successive Effects of the Compounding of Reflexes in the "De-afferented" Condition—Decerebrate Preparations. *Quart. J. of Exper. Physiol.*, 1914, **7**, 407–418.
16. Brown, T. G. Rhythmic Movements as Immediate Reflex Phenomena of Compound Stimulation [Progression Conditioned by Antagonistic Reflex Stimuli]. *Quart. J. of Exper. Physiol.*, 1914, **8**, 155–191.

17. Brown, T. G. Rhythmic Movements as Successive (or Terminal) Reflex Phenomena of Compound Stimulation [Progression Conditioned by Antagonistic Reflex Stimuli]. *Quart. J. of Exper. Physiol.*, 1914, **8**, 193–228.

18. Brown, T. G. On the Phenomenon of Facilitation. 1. Its Occurrence in Reactions Induced by Stimulation of the "Motor" Cortex of the Cerebrum in Monkeys. *Quart. J. of Exper. Physiol.*, 1915, **9**, 81–99.

19. Brown, T. G. On the Phenomenon of Facilitation. 2. Its Occurrence in Response to Subliminal Cortical Stimuli in Monkeys. *Quart. J. of Exper. Physiol.*, 1915, **9**, 101–116.

20. Brown, T. G. On the Phenomenon of Facilitation. 3. "Secondary Facilitation" and its Location in the Cortical Mechanism itself in Monkeys. *Quart. J. of Exper. Phyiol.*, 1915, **9**, 117–130.

21. Brown, T. G. On the Phenomenon of Facilitation. 4. Its Occurrence in the Subcortical Mechanism by the Activation of which Motor Effects are Produced on Artificial Stimulation of the "Motor" Cortex. *Quart. J. of Exper. Physiol.*, 1915, **9**, 131–145.

22. Dittler, R. & Günther, H. Ueber die Aktionsströme menschlicher Muskeln bei natürlicher Innervation, nach Untersuchungen an gesunden und kranken Menschen. *Pflüger's Arch. f. d. ges. Physiol.*, 1914, **155**, 251–274.

23. Eichholtz, F. Ueber das Refraktärstadium im Reflexbogen. Verworn's *Zsch. f. allg. Physiol.*, 1914, **16**, 535–562.

24. Elrington, G. Das Verhalten der Reflexerregbarkeit bei Strychninvergiftung und das "Alles-oder-Nichts-Gesetz." Verworn's *Zsch. f. allg. Physiol.*, 1914, **16**, 115–122.

25. Ernst, Z. Untersuchung über den chemischen Muskeltonus. III. *Pflüger's Arch. f. d. ges. Physiol.*, 1915, **161**, 483–487.

26. Fahrenkamp, K. Ueber die Aktionsströme des Warmblütermuskels im Strychnintetanus. II. *Zsch. f. Biol.*, 1914, **65**, 79–102.

27. Forbes, A. & Sherrington, C. S. Acoustic Reflexes in the Decerebrate Cat. *Amer. J. of Physiol.*, 1914, **35**, 367–376.

28. Forbes, A. & Gregg, A. Electrical Studies in Mammalian Reflexes. I. The Flexion Reflex. *Amer. J. of Physiol.*, 1915, **37**, 118–176.

29. Forbes, A. & Gregg, A. Electrical Studies in Mammalian Reflexes. II. The Correlation between Strength of Stimuli and the Direct and Reflex Nerve Response. *Amer. J. of Physiol.*, 1915, **39**, 172–235.

30. Fredericq, H. Disparition brusque de la conductibilité à la suite d'une compression prolongée ou progressive s'exerçant sur les troncs nerveux. Verworn's *Zsch. f. allg. Physiol.*, 1914, **16**, 213–222.

31. Gildemeister, M. Der sogenannte psycho-galvanische Reflex und seine physikalisch-chemische Deutung. *Pflüger's Arch. f. d. ges. Physiol.*, 1915, **162**, 489–506.

32. Gottschalk, A. Erstickung und Erholung des markhaltigen Kaltblüternerven. Verworn's *Zsch. f. allg. Physiol.*, 1914, **16**, 513–534.

33. Gregor, A. & Schilder, P. Zur Psychophysiologie der Muskelinnervation. *Zsch. f. d. ges. Neur. u. Psychiat.*, 1913, **18**, 195–202.

34. Hoffmann, P. Ueber die Begegnung zweier Erregungen in der Nervenfaser. *Zsch. f. Biol.*, 1914, **64**, 113–124.

35. Hoffmann, P. Zur Frage der Gültigkeit des Alles-oder-Nichtsgesetzes für die Nervenfasern der Krebs- und Hummermuskeln. *Zsch. f. Biol.*, 1914, **64**, 247–262.

36. Hürthle, K. Erwiderung auf die vorliegende Ansicht von Meigs. *Pflüger's Arch. f. d. ges. Physiol.*, 1914, **158**, 100–104.
37. Jensen, P. Weitere Untersuchungen über die thermische Muskelreizung. *Pflüger's Arch. f. d. ges. Physiol.*, 1915, **160**, 333–406.
38. Joel, A. Ueber die Einwirkung einiger indifferenter Narkotika auf die Permeabilität roter Blutkörperchen. *Pflüger's Arch. f. d. ges. Physiol.*, 1915, **161**, 5–44.
39. Kuno, Y. On the Alleged Influence of Adrenaline and of the Sympathetic Nervous System on the Tonus of Skeletal Muscle. *J. of Physiol.*, 1915, **49**, 139–146.
40. Langley, J. N. The Antagonism of Curari and Nicotine in Skeletal Muscle. *J. of Physiol.*, 1914, **48**, 73–108.
41. Lillie, R. S. The Conditions Determining the Rate of Conduction in Irritable Tissues and Especially in Nerve. *Amer. J. of Physiol.*, 1914, **34**, 414–445.
42. Lillie, R. S. The Conditions of Conduction of Excitation in Irritable Cells and Tissues and Especially in Nerve. II. *Amer. J. of Physiol.*, 1915, **37**, 348–370.
43. Lillie, R. S. The General Physico-Chemical Conditions of Stimulation in Living Organisms. *Pop. Sci. Mo.*, 1914, **84**, 579–589.
44. McClendon, J. F. The Action of Anæsthetics in Preventing Increase of Cell Permeability. *Amer. J. of Physiol.*, 1915, **38**, 173–179.
45. Mansfeld, G. & Lukács, A. Untersuchungen über den chemischen Muskeltonus. I. *Pflüger's Arch. f. d. ges. Physiol.*, 1915, **161**, 467–477.
46. Mansfeld, G. Untersuchungen über den chemischen Muskeltonus. II. *Pflüger's Arch. f. d. ges. Physiol.*, 1915, **161**, 478–482.
47. Meigs, E. B. Ob die Fibrillen der quergestreiften Muskeln ihr Volum während der Kontraktion verändern. *Pflüger's Arch. f. d. ges. Physiol.*, 1914, **158**, 92–99.
48. Moldovan, J. & Weinfurter, Fr. Narkose und Sauerstoffatmung. *Pflüger's Arch. f. d. ges. Physiol.*, 1914, **157**, 571–581.
49. Montuori, A. Asfissia e Narcosi. Verworn's *Zsch. f. allg. Physiol.*, 1915, **17**, 18–27.
50. Piper, H. Die Aktionsströme der menschlichen Unterarmflexoren bei normaler Kontraktion und bei Ermüdung. *Arch. f. Anat. u. Physiol., physiol. Abth'g*, 1914, 345–364.
51. Porter, E. L. Variations in Irritability of the Reflex Arc. II. Variations under Strychnine. *Amer. J. of Physiol.*, 1915, **36**, 171–182.
52. Rehorn, E. Das Dekrement der Erregungswelle in dem erstickenden Nerven. Verworn's *Zsch. f. allg. Physiol.*, 1915, **17**, 49–80.
53. Sanders, H.-T. Untersuchungen über die Wärmelähmung des Kaltblüternerven. Verworn's *Zsch. f. allg. Physiol.*, 1914, **16**, 474–505.
54. Schwenker, G. Ueber Danerverkürzung quergestreifter Muskeln, hervorgerufen durch chemische Substanzen. *Pflüger's Arch. f. d. ges. Physiol.*, 1914, **157**, 371–452.
55. Snyder, C. D. Is the Contraction of Smooth Muscle Accompanied by Heat Production? First Communication. *Amer. J. of Physiol.*, 1914, **35**, 340–360.
56. Tashiro, S. & Adams, H. S. Carbon Dioxide Production from the Nerve Fiber in a Hydrogen Atmosphere. *Amer. J. of Physiol.*, 1914, **34**, 405–413.
57. Thörner, W. Ueber den Sauerstoffbedarf des Markhaltigen Nerven. *Pflüger's Arch. f. d. ges. Physiol.*, 1914, **156**, 253–264.
58. Traube, J. Theorie der Narkose. *Pflüger's Arch. f. d. ges. Physiol.*, 1915, **160**, 501–510.
59. Traube, J. Zur Theorie der Narkose. *Pflüger's Arch. f. d. ges. Physiol.*, 1915, **161**, 530–536.

60. TROLAND, L. T. Adaptation and the Chemical Theory of Sensory Response. *Amer. J. of Physiol.*, 1914, 35, 500–527.

61. UEXKÜLL, J. & TIRALA, L. G. Ueber den Tonus bei den Krustazeen. *Zsch. f. Biol.*, 1914, 65, 25–66.

62. WEIZSÄCKER, V. Myothermic Experiments in Salt-Solutions in Relation to the Various Stages of the Muscular Contraction. *J. of Physiol.*, 1914, 48, 396–427.

BOOKS RECEIVED

CRILE, G. W. *Man—An Adaptive Mechanism.* (Ed. by A. Austin.) New York: Macmillan, 1916. Pp. xvi + 387. $2.50.

MÜNSTERBERG, H. *The Photoplay.* New York: Appleton, 1916. Pp. 233.

GATES, A. I. *Diurnal Variations in Memory and Association.* (Univ. of Calif. Publ. in Psychol., 1, No. 5.) Berkeley: Univ. of Calif., 1916.

GATES, A. I. *Variations in Efficiency during the day. Together with Practice Effects, Sex Differences, and Correlations.* (Univ. of Calif. Publ. in Psychol., 2, No. 1.) Berkeley: Univ. of Calif., 1916. Pp. 156.

DEWEY, J. *Democracy and Education.* New York: Macmillan, 1916. Pp. xii + 434.

PATRICK, G. T. W. *Psychology of Relaxation.* Boston: Houghton Mifflin, 1916. Pp. ix + 280. $1.25.

BARTON, W. M. *Manual of Vital Function Testing Methods and Their Interpretation.* Boston: Badger, 1916. Pp. 249.

TUCKER, B. R. *Nervous Children.* Boston: Badger, 1916. Pp. 147.

NOTES AND NEWS

THE present number of the BULLETIN dealing with physiological psychology has been prepared under the editorial supervision of Professor R. P. Angier, of Yale University.

THE following items have been taken from the press:

DR. H. L. HOLLINGWORTH has been promoted to an associate professorship in psychology at Columbia University.

PROFESSOR RAYMOND DODGE has given a course of eight lectures on "Problems and Methods in Dynamic Psychology" at Columbia University.

DR. ERNEST MACH, formerly professor of the history and theory of the inductive sciences at the University of Vienna, has died at the age of 78 years.

ANNOUNCEMENTS of the deaths of Gilbert Ballet, M. Koppen, and Nathan Oppenheim have appeared. Ballet wrote "*Le langage interieur*" and was professor of nervous and mental diseases and the pathology of the brain at the University of Paris. Oppenheim was best known for his books on child hygiene and development. Koppen was professor of psychiatry at the University of Berlin.

DR. LOUIS B. BISCH has been appointed instructor in psychology for the police department, and Dr. Eugene C. Rowe criminologic examiner, of New York City.

PROFESSOR JOSEPH JASTROW, of the University of Wisconsin, recently gave addresses to the Sigma Xi societies at the University of Indiana, on "The Expression of the Emotions," and at Purdue University, on "The Sources of Human Nature," and delivered the Convocation address at the University of Indiana on "Theory and Practice."

ANNOUNCEMENT is made of a prize of $1,000 for the best thesis written by a woman on a scientific subject. Psychology is included among the subjects. The thesis must embody new observations and new conclusions based on independent laboratory research. Theses offered in competition must be in the hands of Dr. Lillian Welsh, Goucher College, Baltimore, Md., before February 25, 1917.